converted into absolute value, as oppose to a gradual rise in the number of Mon2/ml, the number of mDC2/ml remain gradually decreased with the progress of plaque vulnerability. Plasma levels of Lp-PLA2, PTX3, FABP4 and myeloperoxidase, all of which reflected coronary plaque vulnerability, were positively correlated with the number of Mon2/ml, but negatively with the number of mDC2/ml.
Conclusions: Circulating subsets of mDC2 and Mon2 appear to be promosing markers of plaque stabilization and rupture.

GW25-e0795

Relationship between Stress Hyperglycemia and in hospital Mortality and Complications in Patients with Acute Myocardial Infarction

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Objectives: To investigate the effect of stress hyperglycemia on the mortality and cardiovascular events in patients with acute myocardial infarction (AMI).
Methods: The study covered 1226 patients with the diagnosis of acute myocardial Infarction admitted in CCU from January 2010 to December 2012 in the First Affiliate Hospital of Dalian University. Based on the level of fasting blood glucose (FBG), all patients were grouped into two groups: high blood glucose group (HBG group, ≥7mmol/L) and non-high blood glucose group (non-HBG group, <7mmol/L). Based on the history of diabetes were grouped into non-diabetes mellitus group (NDM group) and diabetes mellitus group (DM group). According to the level of FBG, patients within DM and NDM group were further divided into groupA (FBG<7.0mmol/L), groupB (7.0mmol/L≤FBG<9.0mmol/L), groupC (9.0mmol/L≤FBG<11.1mmol/L),groupD (FBG≥11.1mmol/L), respectively. Compare the differences of the mortality and the rate of acute heart failure, cardiogenic shock and main arrhythmias among these groups.
Results: The rate of high FBG is 34.1% in all AMI, among 37% patients without history of diabetes mellitus. Compare with the 808 non-HBG groups patients, the 418 HBG group patients had higher mortality (9.1% vs 2.1%) and higher rate of acute heart failure (37.8% vs 21.2%), cardiogenic shock (7.9% vs 0.7%) and main arrhythmias (25.6% vs 16.5%) (all P<0.01). The mortality (6.4% vs 3.7%) and the rate of acute heart failure (35.9% vs 23%) and cardiogenic shock (6.6% vs 1.7%) are greater in the 362 DM group patients than the 864 NDM group (P<0.05).Among the NDM group, the mortality and cardiovascular complications increased incrementally with the increasing of FBG. Compared with groupA, the mortality and the rate of acute heart failure, cardiogenic shock and main arrhythmias of groupB, groupC, groupD are significantly higher (all P<0.05). Compared with groupB, that mortality of groupD increase obviously (30% vs 2.0%, P<0.01). In the DM group, the mortality showed no significant differences with the increased FBG levels, but the rate of acute heart failure increased incrementally as FBG reached 9 mmol/L compared with group A (P<0.01). The mortality of impaired fasting glucose (IFG) patients is similar to the patients with normal FBG (2.4% vs 1.9%,P>0.05), and significantly lower than the patients with FBG≥7.0mmol/L (11.5% vs 2.4%,P<0.01).
Conclusions: The stress hyperglycemia could be used as a predictor of inhospital mortality and cardiovascular events for patients with AMI. The elevation of FBG increases the mortality and the incidence of acute heart failure, cardiogenic shock, and main arrhythmias of patients with AMI. But the effects were not consistent between DM and NDM patients. In NDM patients, as the FBG level increased, the mortality increased significantly, but this results was not obtained in DM group. In DM group, the incidence of acute heart failure was significantly increased as the FBG level increased.

GW25-e2360

The changes of PPARγ and EPCs in patients with ACS complicated with diabetes mellitus and the effect of Irbesartan on them

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Objectives: To observe the level of PPARγ and EPCs in patients suffered from ACS combined with Type-2 diabetes mellitus (T2DM) and ACS without T2DM, analysis the relationship between PPARγ, EPCs and ACS, T2DM, furthermore explore the influence of Irbesartan on them to study the mechanisms of endothelial protection beyond the antihypertensive effect of Irbesartan, hoping to find a new treatment method for ACS combined with T2DM.
Methods: 102 patients suffered from ACS was enrolled into our study, of which 52 of them combined with T2DM (ACSDM group) and the other 50 patients only suffered from ACS. Meanwhile, we selected 30 patients without coronary heat disease and T2DM as the control group. To all patients, blood was drew when they were enrolled to detect the level of PPARγ and $CD34^+/CD309^+$ EPCs. All basic clinic data, CAG and then Gensini score were compared among all groups. ACSDM group patients were divided into A, B two group randomly. Patients, who were in A group, were treated with Irbesartan 75mg po qd excepting conventional treatment of coronary heart and diabetes. Patients, who wre in B group, were treated without Irbesartan. Blood was drew again after 12 weeks. The level of PPARγ was detected by enzyme-linked immunosorbent method, meanwhile EPCs was detected by Flow cytometry instrument. We analyed datas of each group by applicating of SPSS17.0.
Results: (1) The basic clinic data among each group, such as age, sex, body mass index (BMI), smoking history, blood pressure, stain use, TG, HDL-CH, TC, LDL-CH, blood urea nitrogen, creatinine were no difference (*P* all>0.05). CHD history, STEMI/NSTEMI were no difference in ACS group and ACSDM group (*P* all>0.05). FPG, HbA1c in the ACSDM group were significantly much more than those in the ACS group and control group (*P* <0.05). (2) The levels of PPARγ of ACS group and ACSDM group, when compared with control group (295.56±25.06 ng/L), were decreased significantly (*P* all <0.05), and the ACSDM group was much lower than the ACS group (*P*<0.05). (3) EPCs levels in ACS and ACSDM group were also significantly lower, when compared with control group (0.0584±0.0142%, and the ACSDM group was much lower than the ACS group (*P*<0.05). 4 Gensini points in ACSDM group was much higher than that of the ACS group (42.24±25.46 VS. 30.26±18.35, *P*<0.01). 5 Levels of PPARγ and EPCs were significatly correlated positively (r=0.658, *P*<0.01). Gensini scores of patients with ACS was correlated negatively with both level of PPARγ (r=-0.484,*P*<0.05), and EPCs (r=-0.435,*P*<0.05). Levels of PPARγ and EPCs in combined group were significantly correlated positively (r=0.558, *P*<0.01). 6 After 12 weeks intervention of irbesartan, the level of PPARγ of A group increased significantly compared with pre-treatment levels [(226.17±25.95) ng/L VS. (234.55±26.20) ng/L, *P*<0.01], so did the level of EPCs [(0.0284±0.0111) % VS. (0.0311±0.0102) %, *P*<0.01]. There was a significant positive relationship between them (r=0.767, *P*<0.01). The level of PPARγ and EPCs of B group had on statistical difference after 12 weeks conventional treatment (*P* all>0.05).
Conclusions: The level of PPARγ, EPCs of patients with ACS are siganificantly lower than those of control group, being much lower in ACSDM group. PPAR γ and EPCs level both decrease with the increasing of the degree of coronary artery stenosi. Irbesartan, can improve the levels of PPARγ and EPCs in patients with ACS and T2DM, which probably become a new targets for therapeuty.

GW25-e3089

Bleeding outcomes in low-mediate risk acute coronary syndrome patients receiving stenting predicted by adenosine diphophate induced platelet aggregation after initiation of clopidogrel: 6 months follow-up

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Objectives: The correlation of enhanced platelet responder to clopidogrel with bleeding outcome and entry-site complication has rarely been characterized in China population undergoing percutaneous coronary intervention (PCI). The aim of present study is to access the prognostic significance of optimal platelet inhibition according to a given clinical state and ethnicity.
Methods: A total of 278 non high risk acute coronary syndrome (ACS) patients indicated for PCI were enrolled.Adenosine diphophate induced maximal platelet aggregation (ADP-PG max) was assessed with Lumi-Aggregometer by light transmission aggregometry method. The primary endpoint was the incidence of Thrombolysis in Myocardial Infarction (TIMI) defined bleeding outcome and significant entry-site complication within hospital and 6 months follow-up period. Blood was obtained for platelet aggregation 24h after PCI and 1 month follow up respectively. Receiver-operating characteristic (ROC) curve analysis was conducted to reveal the optimal platelet aggregation value defining enhanced clopidogrel responder for association of measurements with endpoints.
Results: A total of 24 patients (8.6%) met with primary endpoint in the study, while 4 (1.4%) TIMI major bleeding events, 11 (4.0%) minor bleeding events and 9 (3.2%) significant entry-site complications were observed. Follow-up ADP-PG max (OR=0.96, 95%CI, 0.93-0.99; p=0.008) and renal insufficiency (OR=3.38 95%CI, 1.26-9.19; p=0.02) were associate with the prediction of bleeding events. The optimal cutoff for follow up ADP-PG max was 24.5% [area under the curve 0.72 (95% confidence interval 0.59-0.85), p<0.001]. Bleeding occurred in 26.2% of patients with clopidogrel enhanced response (16/61), as compared with 3.7% of remaining patients (8/217), (hazard radio, 9.26; p<0.001).
Conclusions: In conclusion, enhanced clopidogrel responsiveness was associated with a higher risk of bleeding and entry-site complication. Platelet function test detected at an appropriate sampling time after clopidogrel administration may help identify high bleeding risk patients after coronary intervention.

GW25-e3234

Discrepancy in Measuring On-Clopidogrel Platelet Responsiveness by Vasodilator-Stimulated Phosphoprotein Phosphorylation and Platelet Aggregation Is Associated with Smoking Status Following ST-Elevation Myocardial Infarction

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Objectives: To investigate the potential mechanism accounting for the discrepancy of VASP phosphorylation and platelet aggregation (PAG) in evaluating high on-clopidogrel platelet reactivity in patients following ST segment-elevation myocardial infarction (STEMI).
Methods: 90 consecutive STEMI patients scheduled for emergency percutaneous coronary intervention (PCI) were enrolled. Platelet reactivity after clopidogrel loading dose (300 mg) was determined by two methods [platelet reactivity index (PRI), measured by vasodilator-stimulated phosphoprotein (VASP) phosphorylation flow

cytometry and ADP-induced platelet aggregation (PAG), measured by light transmission aggregometry].
Results: All the subjects were dichotomized according to PRI medians (normal-responders: PRI<41.85%, n=45 and low-responders: PRI>41.85%, n=45) and PAG medians (normal-responders: PAG<65.5%, n=49 and low-responders: PAG>65.5%, n=41). No significant differences of demographic data were found between normal- and low-responders determined by the two methods. To further explore the potential clinical characteristics associated with the discrepancy of VASP phosphorylation and PAG in evaluating platelet reactivity, patients who were normal-responders to both methods, or who were low-responders to both methods, were defined as "concordance" group (n=60). Otherwise, the rest patients were defined as "discrepancy group" (n=30). Stepwise binary logistic regression analysis revealed that among factors that potentially influence the consistency of the two methods, smoking was the only independent predictor associated with the discrepancy of the two methods (OR 3.333, 95% CI 1.120 to 9.917, *P*=0.030). After adjusted by the traditional factors (age, gender, hypertension and diabetes) that may influence the responsiveness of platelets to clopidogrel, smoking remains statistically predict the discrepancy between these two methods in evaluating platelet reactivity (OR 4.399, 95% CI 1.105 to 17.522, *P*=0.036).
Conclusions: The present study shows that smoking is an independent factor that influences the consistancy of VASP phosphorylation and ADP-induced PAG in evaluating high on-clopidogrel platelet reactivity in patients with STEMI.

GW25-e4386

The platelet receptor P2Y12 gene polymorphisms and thrombelastography could predict the risk of bleeding events in clopidogrel-treated Chinese patients after percutaneous coronary intervention

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Objectives: Polymorphisms in the platelet receptor P2Y12 gene have been suggested to influence the pharmacodynamics of clopidogrel and prognosis of patients treated with clopidogrel. The effect of P2Y12 polymorphisms and the prognostic utility of thrombelastography (TEG) on bleeding events of clopidogrel treatment have not yet been reported in Chinese patients after percutaneous coronary intervention (PCI). This study sought to investigate the impact of P2Y12 polymorphisms and the predictive value of TEG on the risk of bleeding in clopidogrel-treated Chinese patients after PCI.
Methods: Between January 2011 and June 2012, 504 consecutive patients with acute coronary syndromes (ACS) who received coronary angiography or an uneventful PCI and were exposed to clopidogrel treatment for 12 months, were enrolled in the single-center registry. 18 tag single nucleotide polymorphisms (SNPs) selected from P2Y12 gene and the gain of function CYP2C19*17 allele were detected by the ligase detection reaction. The antiplatelet effect of clopidogrel was assessed by TEG. The primary clinical safety end point was the incidence of major bleeding defined according to the Bleeding Academic Research Consortium (BARC) criteria, including type 3 and 5 in the analysis. The primary clinical efficacy end point was a composite of cardiovascular death, non-fatal myocardial infarction, unplanned target vessel revascularization, and stent thrombosis. The follow-up period was 12 months.
Results: Major bleeding events occurred in 47 patients (9.3%) according to BARC criteria, including 12 patients or 2.4% with BARC type 3b bleeding, 35 patients or 6.9% with BARC type 3a bleeding. By receiver operating characteristic curve analysis, the TEG results of ADP inhibition >80.7% had a predictive value of BARC type ≥3 bleeding with an area under the curve= 0.556 (95%CI 0.511-0.6, p=0.045). Binary logistic regression analysis identified ADP inhibition >80.7% (OR: 2.637, 95%CI: 1.268-5.483, p=0.009), and two tag SNPs rs6785930 (OR: 2.305, 95%CI: 1.180-4.503, p=0.015), rs6809699 (OR: 3.227, 95%CI: 1.587-6.558, p=0.001)) as significant independent predictors of BARC type ≥3 bleeding in the context of gain of function CYP2C19*17 allele. A total of 34 ischemic events occurred. However, no significant influence of tag SNPs of P2Y12 on the occurrence of ischemic events was found. TEG did not predict ischemic events, either.
Conclusions: In our study population, the P2Y12 genetic locus harbors SNPs that could have influence on bleeding events after PCI, and the ADP inhibition of TEG had a predictive value of bleedings.

GW25-e5372

CORRELATION OF NLRP3 WITH PROGNOSIS IN ACUTE CORONARY SYNDROME

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Objectives: To assess whether NLRP3 concentration was related to atherosclerotic severity and any specific features of coronary lesions on angiographic assessment and if NLRP3 concentration has any prognostic value in acute coronary syndrome (ACS) patients.
Methods: In our study we examined one hundred and twenty three (123) subjects including 30 controls (C=30) and 93 Acute coronary Syndrome (ACS=93) patients. We evaluated the association of baseline NLRP3 level with clinical presentation, angiographic characteristics, SYNTAX score, Clinical SYNTAX score (CSS) and Gensini score. Follow up for major adverse cardiac events (MACE) was carried out at 6 months and Kaplan-Meier curves were constructed. Prognostic values of NLRP3, GRACE score and TIMI score were determined by receiver operating characteristic (ROC) curves.
Results: NLRP3 was found to be elevated in ACS patients (P<0.05). There was positive correlation between NLRP3 and SYNTAX score, Clinical SYNTAX score and Gensini score. P value was found to be 0.01 in all cases. NLRP3 was also associated with severity of coronary disease based on number of vessels (P<0.05) involved and the number of lesions (P<0.05) present and also on the presence or absence of bifurcation lesions (P=0.05). The 6 months mortality was 1.07% (1 patient), myocardial infarction 2.15% (2 patients) and unstable angina 3.22% (3 patients), target vessel revascularization was 3.22% (3 patients) and cardiac failure was 4.3% (4 patients). Receiver operating characteristic (ROC) curves of NLRP3 showed good predictive value for MACE.
Conclusions: There are no previous studies related to correlation of NLRP3 levels and severity of atherosclerosis. Our study showed positive correlation between NLRP3 concentration and SYNTAX score, CLINICAL SYNTAX score, GENSINI score, Grace Score and TIMI score. Further studies are required to test the utilization of NLRP3 in risk assessment and in selecting appropriate therapies for patients in low and high risk groups as well as its utilization in decision making regarding the choice of revascularization therapy.

GW25-e1169

Effect of different dose of rosuvastatin on the levels of serum adiponectin, vWF and endothelial function in patients with acute coronary syndrome

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Objectives: To observe and compare the effect of different dose of rosuvastatin on the levels of serum adiponectin (APN), von Willebrand factor (vWF) and endothelial function in patients with acute coronary syndrome (ACS).
Methods: 120 patients with acute coronary syndrome were randomly assigned to receive 10 mg/day or 20 mg /day of rosuvastatin. The levels of serum lipids, adiponectin (APN), von Willebrand factor (vWF) and dilation of the brachial artery were assessed before the therapy, 4 weeks and 8 weeks after the therapy.
Results: All the levels of total cholesterol, low-density lipoprotein cholesterol, triglycerides were significantly decreased from baseline values in Rosuvastatin 10 mg/d and 20 mg/d groups, and the levels of high-density lipoprotein cholesterol increased. After treatment, the level of APN was increased, brachial artery flow-mediated endothelium-depended dilation were improved; while the level of vWF was decreased. These effects were more significant with rosuvastatin for 8 weeks than 4 weeks; but there was no difference between 10 mg/d and 20 mg/d.
Conclusions: Treatment with rosuvastatin 10 mg/d and 20 mg/d could improve abnormal serum lipids and endothelial function, increase APN, decrease vWF. The effect was greater with rosuvastatin for 20 mg/d than 10 mg/d and 8 weeks than 4 weeks.

GW25-e0251

Genetic polymorphisms of CYP2C19*2 and ABCB1 C3435T affect the pharmacokinetics and pharmacodynamics response to clopidogrel in patients with acute coronary syndrome in the Chinese Han population

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Objectives: Clopidogrel is an inhibitor of platelet ADP P2Y12 receptors and plays an important role in the prevention of stent thrombosis. Despite certain clinical benefit using this drug in patients undergoing percutaneous coronary intervention (PCI), some patients do not attain adequate antiplatelet effects. Several studies report that the genetic variation in CYP2C19 and ABCB1 is associated with an impaired response to clopidogrel. This study was designed to investigate the genetic variants of ABCB1, CYP2C9*2, CYP2C19*3, CYP3A4, CYP3A5, P2RY12 and P2Y1 on exposure to clopidogrel (CLP), its active (clopi-H4) and inactive (CLPM) metabolite, and platelet response to clopidogrel in Chinese Han population with acute coronary syndrome.
Methods: The degree of inhibition of platelets was assessed using the light transmittance aggregometry (LTA). Plasma concentrations of CLP, clopi-H4 and CLPM were measured by HPLC-MS-MS method in 401 consecutive patients. Patients were either on 75 mg clopidogrel-maintenance dose or received a 300 mg clopidogrel-loading dose.
Results: In both the treatment groups, carriage of CYP2C19*2 and ABCB1 C3435T was associated with lower exposure to clopi-H4 (P< 0.05) and thus decreased platelet inhibition (P< 0.05), There was no significant association between variant allele PON1 and clopi-H4 formation or antiplatelet response to clopi-H4 in both groups (P> 0.05). CYP2C19*2 is a determinant for the formation of the active metabolite of clopidogrel and its antiplatelet effects. Meanwhile, ABCB1 C3435T also plays an important role in intestinal absorption of clopidogrel, which will further affect the exposure to clopi-H4.
Conclusions: In both the treatment groups, carriage of CYP2C19*2 and ABCB1 C3435T was associated with lower exposure to clopi-H4 (P< 0.05) and thus decreased platelet inhibition (P< 0.05), There was no significant association between variant allele PON1 and clopi-H4 formation or antiplatelet response to clopi-H4 in both groups (P> 0.05). CYP2C19*2 is a determinant for the formation of the active